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Infiltration of inflammatory cells in intestines of chickens infected naturally by *Ascaridia galli*

[1*]Muhammad Hambal, [2]Ummu Balqis, [3]Darmawi, [1]Cut S. Utami

[1]Laboratory of Parasitology, Faculty of Veterinary Medicine of Syiah Kuala University, Banda Aceh 23111, Indonesia;

[2]Laboratory of Pathology, Faculty of Veterinary Medicine of Syiah Kuala University, Banda Aceh 23111, Indonesia;

[3]Laboratory of Microbiology, Faculty of Veterinary Medicine of Syiah Kuala University, Banda Aceh 23111, Indonesia;

*Coorresponding Author: umm1334@yahoo.com

Abstract. This research was carried out to investigate the infiltration of inflamatory cells in the intestines that were naturally infected by *Ascaridia galli*. Ten intestines were obtained from wet market in Banda Aceh. Inflamatory cells were assayed by in situ jejunal mast cell counts in stained histological sections of the duodenum, jejunum and ileum, of which histologic slides were made. Duodenum, jejunum and ileum infected by *Ascaridia galli* showed hyperemia, and inflammatory cell infiltration in part of A. galli infection.

Keywords: Intestine, chicken, *Ascaridia galli*

Introduction

Nematode infection in the small intestine causing inflammatory cell infiltrate composed of lymphocytes, eosinophils and macrophages. Inflammatory cell infiltration caused by the body's attempt that seeks to remove the antigen. Leukocyte cell functions provide a fast and strong defense against any pathogen that enters the body (Guyton, 1996). According to the observations Riwidiharso (1989), recurrent infections of Ascaridia galli generate immunity in chickens characterized by an increase in the number of leukocytes, especially lymphocytes. Therefore, we investigated the distribution of inflamatory cells in naturally *A. galli*-infected chickens under histological conditions.

Materials and Methods

Intestine was dissected, flushed with cold sterile saline solution, opened longitudinally, and placed, mucosa side up, onto small pieces of blotting paper. The segments were then fixed in 10% buffered normal formalin. This process was performed for each leying hen using sterile instruments for each dissection. Fixed samples were dehydrated in the ascending concentrations of ethanol (50%, 60%, 70%, 80%, 96% (1), 96% (2) and 100%). The samples were cleared in xylol and were embedded in paraffin wax. Serial histological sections (3-5 µm of thickness) were stained with Hematoxylin Eosin as described by Darmawi *et al*. (2012) with certain modifications.

Results and Discussion

The numbers imflamatory cells varied widely and they distributed within the layer of intestines (Figure 1). In the intestinal tract of infected laying hens, inflamatory cells were found in lamina propria mucosae tissue layers. We described that laying hens infected naturally by *A. galli* accumulated inflammatory cells in the intetinum. Increased numbers of inflammatory cells are often observed in affected tissues during helminth infections.

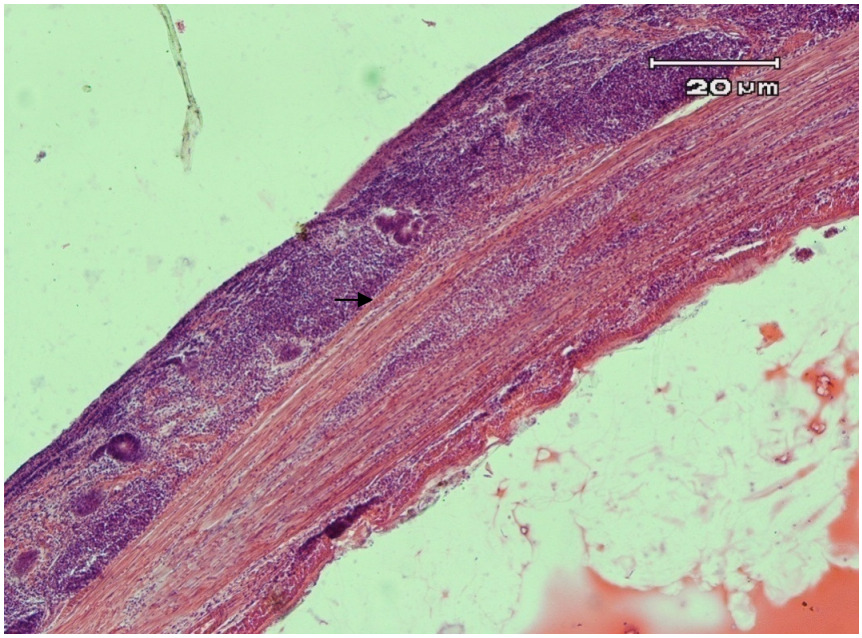

Figure 1. Picture of inflammatory cell infiltration chicken intestines were naturally infected with *Ascaridia galli*. Arrows (→) indicate infiltration of inflammatory cells (HE, 100x)

Based on Figure 1 shown that inflammatory cells dominate and form the nest. A significantly higher number of eosinophils were found in lamina propria of the layer pullets seven-week old at three days after infected orally with 20,000 embryonated *A. galli* eggs group compared to the control group (Luna-Olivares *et al*., 2012). Else and Finkelman (1998) explained that immue expulsion of adult *T. spiralis* worms parasitizing the small intestine is a complex process ascociated with a Th2 mediated eosinophilia, goblet cell hiperplasia, and mastocytosis. The villous stroma of the duodenum of albino rats infected by *Centrocestus armatus* was moderately infiltrated by inflammatory cells such as lymphocytes, plasma cells, and eosinophyls, and that of jejunum was moderately udematous (Hong *et al*., 1997). Ierna *et al*. (2005) have shown that the intestinal inflammation, which is controled by IL-4, is not required for parasite expulsion. The OX40-OX40 ligand to be important for development of Th2 immune responses, but also involved in a number of imflammatory disease, including those of the intestine. The immune expulsion of gastrointestinal parasite is usually ascociated with Th2 responses. Koski and Scott (2003) Gastrointestinal nematode, such as hookworms, *Ascaris lumbricoides*, and *Thricuris trichiura*, require activation of the Th2 arm of the immune cascade and expresion

of the Th2 cytocine (IL-4, IL-5, and IL-10), and their effectors for worm expulsion. At the cytoplasmic granules of eosinophils are small granules that contain histaminase, proteins such as peroxidase, RNase, DNase, lipase, plasminogen and Major Basic Protein (Major Basic Protein) which is toxic to both parasite and host tissues (Zalizar et al., 2006). On worm infection, eosinophil cells move to the site of infection to kill the worms. Eosinophil cells migrate into the infected area and releasing parasite enzymes destroyer or destroyer of parasites. Macrophage phagocytosis and effort involved in antigen processing in preparation for the reaction of antibody-mediated immune response and cellular immune response (Tizard, 1982).

Conclusions

Based on the results of this study concluded that the chicken (*Gallus domesticus*) infected naturally by Ascaridia galli caused the infiltration of inflamatory cell and hemorrhagy in the intestine.

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